CASE REPORT

Patent Ductus Arteriosus in a Cat

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Summary

Patent ductus arteriosus is the most common shunting congenital cardiac anomaly in the cat. If it is treated surgically, early in the course of the disease, the prognosis is good.

Résumé

Persistance du canal artériel, chez un chat

La persistance du canal artériel correspond à l'anomalie cardiaque dérivative congénitale la plus fréquente, chez le chat. Une intervention chirurgicale précoce permet de donner un pronostic favorable.

Introduction

The incidence of congenital and acquired feline cardiac disease, although reported to be less common than that observed in the canine and human, is likely more prevalent than was previously believed (4,10,15). Feline heart disease is seen in only 1: 1 000 cases compared with 94: 1 000 cases for canines (4,10). These figures are probably an underestimation because kittens and pups with severe congenital cardiac lesions usually die before weaning and are never necropsied (10). Patent ductus arteriosus (P.D.A.) is the most common congenital heart disease observed in cats (10). This paper reports such a clinical case and discusses the disease.

History and Clinical Findings

A four month old, domestic long hair male cat was referred to the Ontario Veterinary College with a history of wheezing, poor exercise tolerance and ascites. The onset of these problems was noticed at four weeks of age.

A general physical examination revealed a thin but normal sized kitten, as compared to its littermates. The respiratory rate was increased and moist rales were heard on auscultation. Ascites was also present. A machinery murmur, grade V/VI, loudest over the left hemithorax near the cardiac base was auscultated, and a strong thrill was palpated over the same area. The pulse was weak.

A complete blood count, biochemical profile, chest radiograph and electrocardiogram (ECG) were performed. The ECG revealed biventricular enlargement, left atrial enlargement and first degree heart block. Thoracic radiographs demonstrated generalized cardiac enlargement.

Diagnosis and Treatment

On the basis of the history, physical examination and ancillary tests, a diagnosis of cardiac disease due to patent ductus arteriosus was made. Surgery for repair of the patent ductus arteriosus was performed in a routine manner as previously described (2,6,13). Recovery was uneventful and the kitten was discharged six days postoperatively.

Discussion

The ductus arteriosus arises from the left sixth aortic arch in fetal development, to circumvent the pulmonary circulation (1). The P.D.A. enters the pulmonary trunk just anterior to the bifurcation of the left and right pulmonary arteries. The ductus usually closes about the second week postpartum in the dog, by a process of intimal proliferation, smooth muscle degeneration, and fibrosis, to form the ligamentum arteriosum (1). The stimulus to close is likely a combination of hemodynamic, neurological, humoral and gas tension effects initiated with the onset of respiration and a fall in pulmonary vascular resistance (5,7). It has been shown that the smooth muscle fibers of the ductus arteriosus will contract in the presence of oxygen levels normally found in arterial blood. When the animal takes its first breath the pulmonary vascular resistance drops as the lungs expand. Then there is a reversal of blood flow in the ductus arteriosus and a consequent rise in the oxygen tension as blood flows from the aorta to the pulmonary artery via the ductus arteriosus. Respiratory difficulties at birth have been associated with incomplete closure (7,8).

Patent ductus arteriosus appears with greater frequency in female dogs. It is genetically transmitted in family lines, but phenotypically normal animals may transmit the condition to their offspring. In the cat, P.D.A. is seen most often in the Siamese (10,15). The pulmonary trunk is dilated and intimal fibrosis caused by the jetting of blood flow is seen in the lumen.

Initially, decreased systemic circulation caused by the shunting, causes the release of aldosterone and antidiuretic hormone. A subsequent retention of fluid results in an increase in venous return and concomitant increase in cardiac output. The left ventricle, in order to expulse this increased blood volume, enlarges and hypertrophies. Pulmonary hypertension and increased right ventricular pressure may lead to right ventricular enlargement as well (10).

Affected cats tend to sleep more, have poor exercise tolerance, pant after exercise and sometimes cough. Their growth may also be somewhat retarded. Owners may relate about feeling a buzzing in the pet's chest when they pick the cat up. Affected cats tend to develop signs of heart failure at an average of five months of age (4,13). The physical examination is generally quite unremarkable. Cyanosis of the posterior portion of the body, if seen, occurs only late in the course of

the disease. Cyanosis can be seen if pulmonary hypertension develops causing a reversal of blood flow. Oxygen-poor blood then flows from the pulmonary artery to the aorta. This will be especially noticeable after stress or exercise. Heart rates tend to be rapid. The pulse has been described as a waterhammer, "BB" shot or jerky. If reflects the wide pulse pressure (1,5,13). The pulse collapses quickly during diastole due to the run off of blood of the pulmonary artery.

A continuous, machinery murmur, is heard best over the left hemithorax in the area of the pulmonic valve. The intensity of the murmur increases with the onset of the second heart sound and is inversely proportional to the size of the ductus arteriosus in dogs (11). Hence the murmur may be absent in large ductal lesions. It may also decrease or become systolic in nature with the onset of pulmonary hypertension and the reversal of blood flow.

Electrocardiograms may show left ventricular enlargement by the presence of tall R waves in leads 11, 11, and 111 and left atrial enlargement by the prolonged P waves in the dog (1,5,13).

Radiographs may reveal a bulge of the aorta, main pulmonary artery and left auricle on the dorsoventral view. Often only generalized cardiomegaly and pulmonary edema is identified (5,13).

Cardiac catheterization demonstrates an increased oxygen tension in the pulmonary artery and normal or elevated right heart pressures if pulmonary hypertension develops (10). Angiography will show a simultaneous outlining of the aorta and main pulmonary artery after selective left heart injection of contrast (5,13).

The treatment is surgical ligation of the patent ductus arteriosus. If left untreated, most patients die from heart failure and pulmonary edema before adulthood. The P.D.A. results in left ventricular dilatation and mitral insufficiency with subsequent left heart failure. In one study in dogs, the death rate of untreated cases was 64% versus 8% of those treated surgically (6). Best results are obtained in the young patient before permanent cardiac changes have occurred. Surgery is contraindicated if a reversal of blood flow has occurred as ligation of the P.D.A. would cause massive overcirculation in the lungs and death. Reversed flow may be demonstrated by cardiac catheterization or suggested clinically by the occurrence of cyanosis and the loss of the machinery murmur.

The surgical approach and corrective technique have been well documented elsewhere (2,13). The prognosis postoperatively is good in uncomplicated cases. The cardiac silhouette can be expected to decrease in size. The ECG will also return to normal values. A systolic heart murmur may remain though, because of the stretching of the mitral annulus and mitral insufficiency.

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ABSTRACT

Fisher, E.W. Neonatal survival. British Veterinary Journal (1980) 136: 585-589. (Dep. Vet. Med., Univ., Glasgow, UK).

Recent work is reviewed, with particular reference to the role of immune globulins. A simple turbidity test showed that calves with minimal readings died from septicaemia in the first week of life, and those with low readings died from diarrhoea and wasting but without PM signs of septicaemia. Calves with high readings survived. Deaths from septicaemia were related to deficiency of IgM globulins, and those from diarrhoea to IgA deficiency. Survival depended on high IgA plasma concentrations. Poor globulin absorption was probably due to feeding insufficient colostrum or feeding it too late. It is recommended that cow and calf be left undisturbed for the first 12 hours. Survival in lambs, piglets, foals and puppies is also discussed.

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